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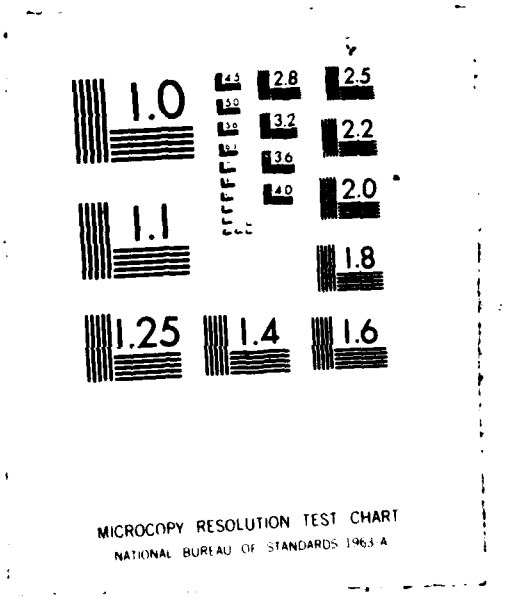
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EFFECT OF AEROBIC TRAINING ON THE PLASMA ACTH RESPONSE TO EXERCISE

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EFFECT OF AEROBIC TRAINING ON THE PLASMA ACTH RESPONSE TO EXERCISE

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Summary

A previous study has shown that plasma adrenocorticotropin (ACTH) secretion during submaximal exercise is increased following training. This finding is in contrast to studies of other hormones such as glucagon, epinephrine and growth hormone, all of which show decreases following training. This study re-examines the effect of training on plasma ACTH levels during exercise. Ten adult volunteers were split into a control and an exercise group. The exercise group underwent a 12 week training program consisting of running 3.5 kilometers per day, three days per week. Before and after the training program all subjects were tested for plasma ACTH response to a 150 Watt workload for 15 minutes. The experimental group showed an 11% increase in Max Vo_2 following training while the control group's Max Vo_2 was unchanged. The experimental group's ACTH response to the submaximal workload was decreased 63% ($p<0.05$), while the control group's response was unchanged. These data suggest that the ACTH response to exercise is blunted following training. This response is similar to that of other hormones such as glucagon, epinephrine and growth hormone. Such a finding is in agreement with previous studies that have shown that ACTH follows an intensity dependent pattern during exercise.

Introduction

A review of the literature (3,6) reveals that only one study (1) has previously measured plasma adrenocorticotropin hormone (ACTH) during exercise in humans before and after a physical training program. The results of that study (1) suggest that training augments the secretion of ACTH in response to a sub-maximal exercise bout.

Such a finding is interesting in light of two facts. First, most studies report that exercise training usually results in reduced plasma levels of glucagon, epinephrine, norepinephrine, and growth hormone (3,6) in response to the same absolute workload. Thus, the study by Carr et al (1) is not in agreement with these previous findings. Second, most studies (6) report that at the same sub-maximal workload trained individuals have a smaller rise in plasma cortisol than do untrained individuals. Since cortisol secretion is under the control of ACTH, it is difficult to explain how training decreases the cortisol response to exercise while at the same time increasing plasma ACTH levels. Therefore, in light of the above discussion, it was the purpose of this study to re-examine the effect of training on plasma ACTH levels during exercise.

Methods

The subjects for this study were 10 adult volunteers (4 male, 6 female). Written informed consent was obtained from each subject prior to testing.

The 10 subjects were split into a control group and an experimental group, each of which contained 2 males and 3 females. Each subject reported to the laboratory on 4 separate occasions. On the first visit each subject performed a graded treadmill run to exhaustion. Oxygen uptake was measured each min of the run using open-circuit spirometry (Alpha-Technologies, System 4400, Laguana Hills, CA) with the highest value being recorded as maximal O_2 uptake (VO_2 max).

On the second visit, the subjects rested in a seated position for 10 min, following which a 10 ml venous blood sample was obtained from an antecubital vein. Each subject then exercised on a Godart cycle ergometer at 150W for 15 min. A second venous blood sample was obtained immediately upon completion of the exercise bout.

Following the second lab visit the subjects in the experimental group started a 12-week exercise training program. The subjects ran 3.5 km a day, three days a week for the 12 week period. The subjects' exercise heart rate during the training runs was held between 70-85% of their previously determined maximal heart rate. The control group did not participate in the organized exercise sessions. Furthermore, they were instructed not to change their exercise habits over the course of the study.

Following the 12 week training program all of the subjects reported to the laboratory for their third lab visit and again performed a graded treadmill run to exhaustion. On their fourth visit

they repeated the sub-maximal exercise protocol that they performed on their second visit. The second and fourth lab visits were held at approximately the same time of day for each subject.

All of the collected blood samples were measured, in duplicate, for plasma ACTH using the RIA procedures described by Orth (11). Intra-assay coefficients of variation were 3 and 5% respectively, at 80 and 20% B/B_o. The working standard ACTH reference preparation for the assay was synthetic human ACTH (1-39). The sensitivity of the assay was 5 pg/ml.

Independent t-tests were used to test for differences between the two groups. Paired t-tests were used to examine the effect of training on VO_2 max and ACTH. Significance was set at the $p \leq 0.05$ level.

Results

The mean ($\pm$S.E.) descriptive and fitness variables for the two groups are shown in Table 1. As can be seen, there was no significant difference between the two groups in age, ht, wt, or pre-training VO_2 max. The training program, however, produced a significant increase in VO_2 max in the experimental group, while the control group did not change significantly over the 12 week period. The mean 11% increase in VO_2 max in the experimental group is similar in magnitude to previously reported training studies (12).

Table 1

Descriptive Variables for the Experimental and Control Groups Before and After Training

	Age (Yrs)	HT(cm)	Wt(kg)	VO2Max pre($Lmin^{-1}$)	VO2Max post($Lmin^{-1}$)
Experimental (n=5)	22.1$\pm$0.4	165.6$\pm$1.3	67.6$\pm$1.5	2.98$\pm$.28	3.20$\pm$.36*
Control (n=5)	26.6$\pm$0.8	168.0$\pm$1.1	61.7$\pm$2.0	2.70$\pm$.24	2.78$\pm$.23

Values are mean $\pm$S.E.

*pre- vs post-training values are significantly ($p \leq 0.05$) different

The exercise induced plasma ACTH response for both groups is shown in Fig. 1. As can be seen, the experimental group demonstrated a significant reduction in the delta ACTH response to the 150W exercise bout following the training program. Delta ACTH is simply the post-exercise ACTH value minus the pre-exercise value, and represents the increase in plasma ACTH that resulted from the exercise bout. The control group did not show a significant difference in the delta ACTH response to the exercise bout over the course of the study.

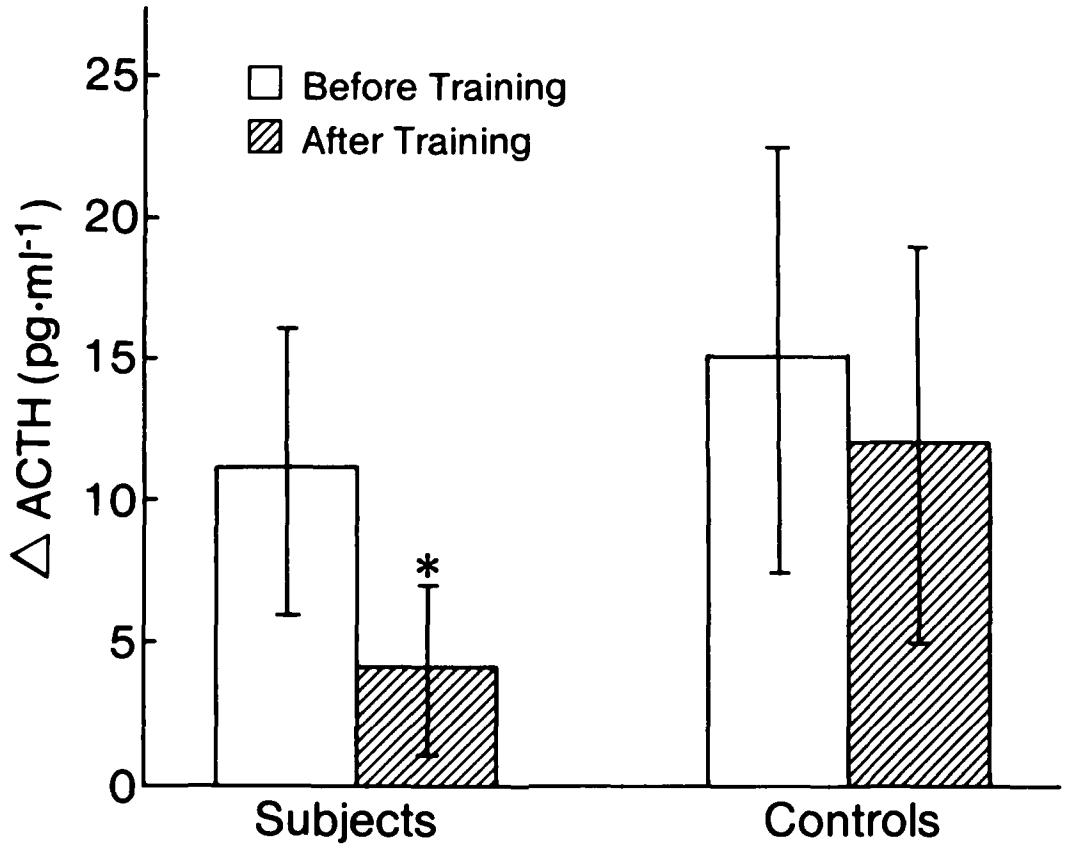

Fig. 1. Mean (±S.E.) delta ACTH responses for the two groups before and after the training program.

Discussion

The most important finding of the current study is that the ACTH response to sub-maximal exercise is blunted following a training program. This finding suggests that plasma ACTH responds in a similar fashion to training when compared to many other hormones (3,6). The results of this study, however, are in conflict with the previously published work of Carr et al (1). They found that ACTH levels rose 63% during exercise prior to training and by 116% after two months of aerobic conditioning. A closer examination of their paper, however, reveals that the workloads used to elicit the ACTH response increased from a mean of 70W to 100W during the course of the study. From this data it can be estimated that their subjects would have had to have increased their VO_2 max over the course of the study 32% to have made the 70W and 100W workloads of equal relative intensity. As suggested by Galbo (6), it seems more likely that the workloads over the course of the study made up an increasing percentage of the subjects VO_2 max. If the above assumption is correct then the data of Carr et al (1) is consistent with the findings of Farrell et al (4) who have reported that ACTH increases during exercise in an intensity dependent pattern.

In the current study the 150W workload represented a mean (±S.E.) of 73±6% of the experimental groups pre-training VO_2 max but only 66±7% of their post-training VO_2 max. These results also support the previously determined intensity dependent pattern of ACTH during exercise (4).

The mechanism for the blunted ACTH response to exercise following training can only be speculated on at this time. However, at least 3 potential explanations seem worthy of discussion. First, previous studies (9) have shown that arginine vasopressin (AVP) can potentiate the effect of hypothalmic corticotropin releasing factor (CRF) on ACTH secretion. Interestingly, it has been reported (2) that training reduces the AVP response to sub-maximal exercise. Thus, the decreased ACTH response to exercise following training may be secondary to a decrease in AVP.

Second, previous investigators (4,5) have suggested that disturbances in intracellular homeostasis during exercise may be sensed by peripheral chemoreceptors which stimulate ACTH secretion. Such a hypothesis is consistent with the results of the current study, since it is known that following training the intracellular homeostasis is less disturbed, as evidenced by smaller changes in muscle lactate and phosphagen levels (10) at the same absolute workload.

Lastly, several animal studies at least suggest that cardiovascular dynamics during exercise may act as a stimulus to ACTH release (8). In the current study the mean (±S.E.) exercise heart rate in the experimental group during the 150W workload did decrease significantly from 154 ± 13 bpm to 146 ± 13 bpm. However, there was not a significant correlation ($r = -.034$, NS) in the experimental group between the delta ACTH and delta exercise heart rate responses pre- to post-training. Such a finding is in agreement with others (4,7) who also did not demonstrate a significant relationship between heart rate and ACTH.

In conclusion, the current study shows that the ACTH response to a sub-maximal workload is blunted following training. Such a finding is in agreement with previous work that has shown that ACTH follows an intensity dependent pattern during exercises.

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The purpose of this study was to re-examine the effect of training on plasma ACTH levels during exercise. Ten adult volunteers were split into a control and an experimental group. The experimental group participated in a 12-week training program which resulted in a significant 11% increase in their mean maximal oxygen uptake. The plasma ACTH response to a 150W workload was measured in both groups before and after the training program. The experimental group demonstrated a significant reduction in the delta ACTH response to the workload, while the control group demonstrated an unchanged response over the course of the study. These data suggest that the ACTH response to an absolute, submaximal workload is blunted following training. Such a finding is in agreement with previous studies that have shown that ACTH follows an intensity dependent pattern during exercise.

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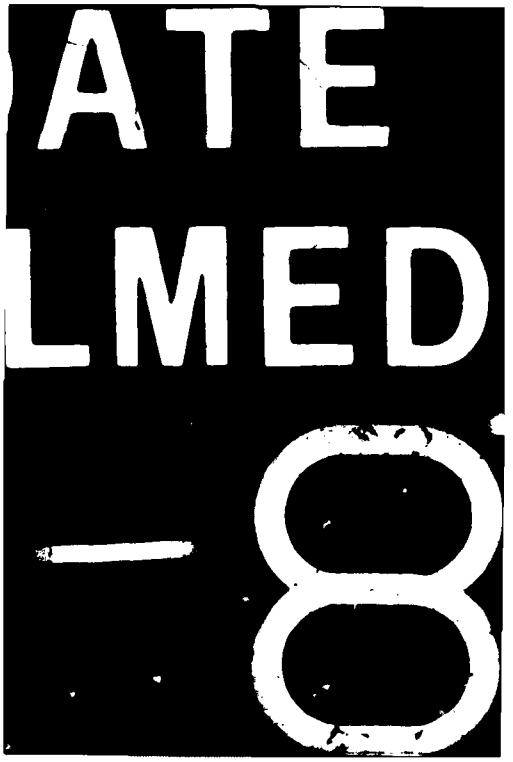
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